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Effects of intermittent pneumatic foot compression on blood coagulability and fibrinolysis assessed by a whole blood viscometer Sonoclot

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Perioperative pulmonary embolism is an infrequent but potentially catastrophic complication. Intermittent pneumatic compression (IPC) is a useful method for prophylaxis of perioperative venous thromboembolism. Although its efficacy has been shown to depend on both inhibition of blood congestion and stimulation of endogenous fibrinolytic activity [1–4], its overall effect on blood coagulability has not been reported. We hypothesized that IPC-generated shear force alters platelet function and/or coagulation/fibrinolysis factors derived from endothelium. Mechanical IPC devices to prevent deep vein thrombosis are currently available in two forms: a standard low-pressure, slow-inflation device (SeQuel; Kendall, Mansfield, MA, USA) and a high-pressure, rapid-inflation device (AV impulse; Norvamedix, Hampshire, UK). The aim of the present study was to clarify the effects of these two kinds of IPC device on whole blood coagulability and fibrinolysis using a blood viscometer (Sonoclot; Scienco, Wheat Ridge, CO, USA).

After obtaining approval for our study from our institutional ethics committee on human research and informed consent from the subjects for participation in the study, 12 adult volunteers [mean (± SD) age 30 ± 5 years, height 168 ± 12 cm, weight 68 ± 7 kg] were enrolled. All of the subjects were graded as ASA 1 (without any systemic disease), and none of the subjects had obesity (body mass index > 28), vascular disease, or abnormal coagulopathy. They were randomly (by the envelope technique) divided into two groups according to the type of IPC device used: standard or rapid. The subjects were requested to lie in a supine position and to remain calm. Both soles of each subject in one group were compressed for 3 s (130 mmHg) at 0.3-Hz intervals during rapid IPC. In the other group, both legs of each subject from the ankles to the thighs were compressed by the standard IPC device, which has a six-chambered cuff that applies compression of 45 mmHg for 12 s sequentially followed by a 60-s noncompression period. To measure whole blood coagulability using the Sonoclot viscometer, venous blood samples were collected from the left cephalic vein at 10 min before compression (control) and at 25 and 60 min after compression. The following Sonoclot variables were recorded: sonACT, i.e., Sonoclot-measured activated clotting time (liquid phase or onset of clot formation); clot rate, i.e., slope of the Sonoclot signature (fibrin gel formation stage); and time-to-peak, i.e., duration from the start of measurement to the peak of the Sonoclot signature (Fig. 1A). Data are expressed as means ± SD. All data were analyzed by one-way ANOVA, and Fisher's test was used as a post hoc test. $P < 0.05$ was considered to indicate statistical significance.

Typical time courses of the Sonoclot signature before and 25 min after IPC by the use of the AV impulse are shown in Fig. 1. The AV impulse significantly shortened the time-to-peak without changing sonACT or clot rate. Both of the IPC devices significantly shortened the time-to-peak in a time-dependent manner (by more than 40% at 60 min after compression). The other parameters, sonACT and clot rate, were not changed during the study period by the use of either device (Table 1). There were no significant differences in these parameters between the IPC devices tested.

The Sonoclot analyzer is used as a blood viscoelastic monitor. One of the benefits of its use is that the Sonoclot signature provides global measurement of

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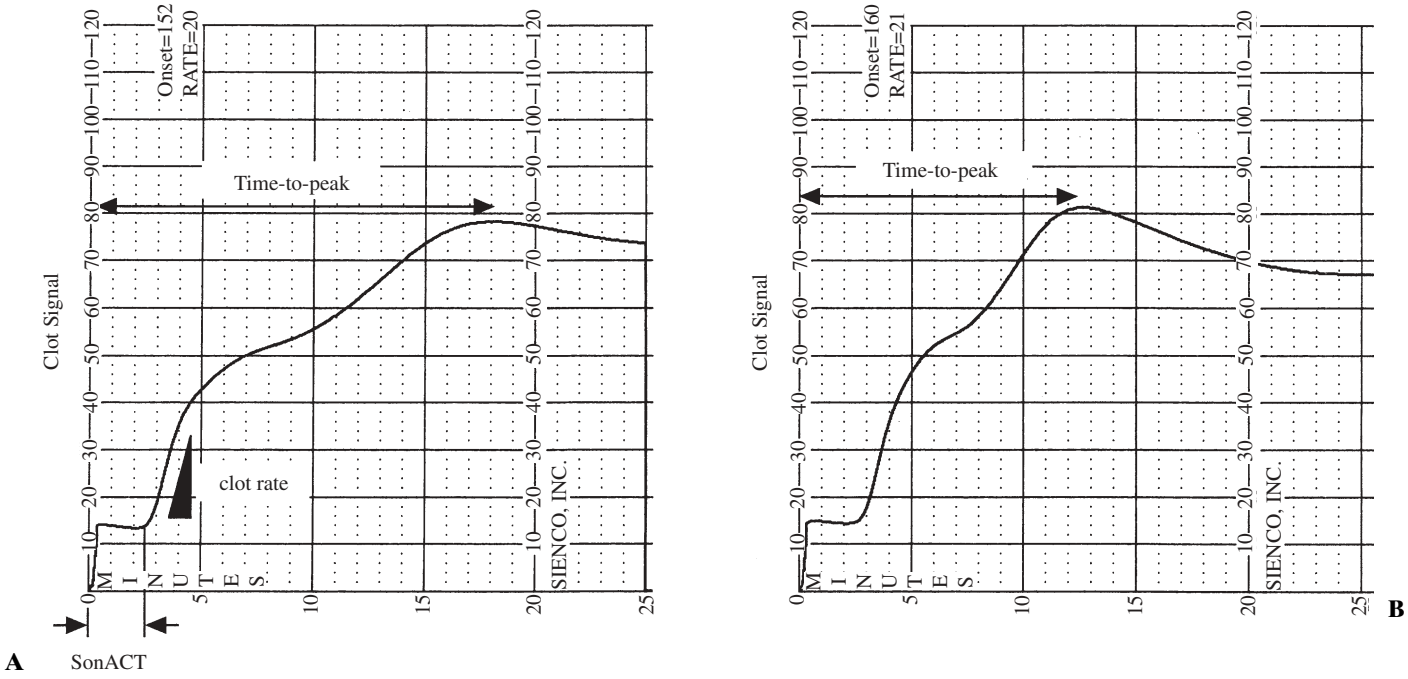

Fig. 1. Typical traces of Sonoclot signature during the study period by use of AV impulse. **A** Control. *SonACT*, Sonoclot-measured activated clotting time; *clot rate*, slope of the Sonoclot signature; *time-to-peak*, duration from the start of measurement to the peak of the Sonoclot signature. **B** 25 min after compression by AV impulse

Table 1. Changes in measured parameters during the study period

Parameter	Control	25 min	60 min
AV impulse			
SonACT	128.3 ± 33.0	154.8 ± 18.3	135.5 ± 38.5
Clot rate	16.8 ± 5.0	20.2 ± 4.5	19.5 ± 2.7
Time-to-peak	16.2 ± 5.1	12.4 ± 4.7*	7.3 ± 4.9**†
SeQuel			
SonACT	145.2 ± 24.9	126.0 ± 31.1	126.2 ± 32.0
Clot rate	18.2 ± 4.0	16.3 ± 2.4	16.7 ± 3.1
Time-to-peak	18.1 ± 6.2	15.4 ± 5.3	9.2 ± 3.9**†

Means ± SD, $n = 6$ each
* $P < 0.05$, ** $P < 0.01$ vs control, † $P < 0.05$ vs 25 min
SonACT, Sonsoclot-measured activated clotting time; clot rate, slope of the Sonoclot signature; time-to-peak, duration from the start of measurement to the peak of the Sonoclot signature; 25 and 60 min, 25 min and 60 min after compression by the intermittent pneumatic compression devices used

hemostasis, including plasma coagulation, platelet function, and retraction/fibrinolysis [5]. The results of the present study showed that IPC induced a significant shortening of the time-to-peak parameter in Sonoclot traces. Time-to-peak can be determined by the balance between platelet activation and clot retraction/fibrinolysis [6], and its value is generally thought to reflect platelet function. The manufacturer of Sonoclot and several investigators have pointed out that an inadequate number of platelets or poor platelet function is often associated with a prolonged time-to-peak on the Sonoclot signature [5]. Thus, it seems possible that IPC shortens the time-to-peak parameter in the Sonoclot signature by enhancing platelet activity. However, this possibility can be ruled out as a mechanism for time-to-peak shortening, because other coagulation parameters, such as SonACT and clot rate, did not change [6]. Another possibility for the mechanism of time-to-peak shortening is activation of fibrinolysis, because activation of fibrinolysis can also shorten the time-to-peak. Some studies have shown that stimulation of endogenous fibrinolytic activity occurred after IPC with a resulting increase in tissue-type plasminogen activator (t-PA) activity in vivo [3] and in vitro [7]. Dai et al. [7] showed by using messenger RNA analysis that IPC induces an up-regulation of tPA and endothelial nitric oxide synthase expression. Based on these findings, it can be speculated that increased tPA activity and/or nitric oxide level induced retraction of the clot and shortening of the time-to-peak. Dai et al. [6] also demonstrated that pulsatile flow, more than vessel compression, influenced endothelial function. However, further study, such as measurement of tPA activity, is needed to clarify this point.

In conclusion, we investigated the effects of using two kinds of IPC device, AV impulse and SeQuel, on overall blood coagulability and fibrinolysis, as assessed by the use of a viscometer (Sonoclot). Both of the IPC devices shortened the time-to-peak parameter without causing

changes in the other coagulation parameters, sonACT and clot rate. Although time-to-peak shortening can be caused by an increase in fibrinolytic activity, further study is needed to elucidate the effects of IPC on overall coagulability and fibrinolysis.

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